Search:
US 5976806 (Mahajan et al. -DNA ligase assay)
BRS L1 4 dna adj repair adj ligase USPAT 2009/05/21 13:40

IS&R L2 1 ("20070172822").PN. US-PGPUB; USPAT; USOCR 2009/05/21 13:46
BRS L4 0 prokaryotic adj dna adj ligase USPAT 2009/05/21 13:50

BRS L5 13503 dna adj ligase USPAT 2009/05/21 13:52
BRS L7 7 l6 and prokaryotic.clm. USPAT 2009/05/21 13:53
BRS L6 190 (dna adj ligase).clm. USPAT 2009/05/21 13:52
BRS L8 158 l6 and method.clm. USPAT 2009/05/21 14:05
BRS L9 0 l8 and ligaase.ti. USPAT 2009/05/21 14:06
BRS L10 3 l8 and ligase.ti. USPAT 2009/05/21 14:06
BRS L11 40 l8 and (prokaryotic or procaryotic) USPAT 2009/05/21 14:12

STN:

(FILE 'HOME' ENTERED AT 15:14:01 ON 21 MAY 2009)

FILE 'MEDLINE, BIOSIS, BIOTECHNO, CAPLUS, EMBASE, JAPIO' ENTERED AT 15:14:47 ON 21 MAY 2009
L1 28 S DNA REPAIR LIGASE
L2 24 DUP REM L1 (4 DUPLICATES REMOVED)
L3 10522 S DNA LIGASE
L4 34 S L3 AND MYCOBACTERIUM AND COLI
L5 18 DUP REM L4 (16 DUPLICATES REMOVED)

L5 ANSWER 13 OF 18 MEDLINE on STN DUPLICATE 5
AN 2003177363 MEDLINE
DN PubMed ID: 12696044
TI NAD+-dependent DNA ligases of Mycobacterium tuberculosis and Streptomyces coelicolor.
AU Wilkinson Adam; Sayer Heather; Bullard Desmond; Smith Andrew; Day Jonathan; Kieser Tobias; Bowater Richard
CS Phico Therapeutics, Ltd., Babraham Hall, Babraham, Cambridge, United Kingdom.
SO Proteins, (2003 May 15) Vol. 51, No. 3, pp. 321-6.
Journal code: 8700181. E-ISSN: 1097-0134.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200306
ED Entered STN: 17 Apr 2003
Last Updated on STN: 17 Jun 2003
Entered Medline: 16 Jun 2003
AB Sequencing of the genomes of Mycobacterium tuberculosis H37Rv and Streptomyces coelicolor A3(2) identified putative genes for an NAD(+)-dependent DNA ligase. We have cloned both open reading frames and overexpressed the protein products in Escherichia coli. In vitro biochemical assays confirm that each of these proteins encodes a functional DNA ligase that uses NAD(+) as its cofactor. Expression of either protein is able to complement E. coli GR501, which carries a temperature-sensitive

mutation in ligA. Thus, in vitro and in vivo analyses confirm predictions
that ligA genes from M. tuberculosis and S. coelicolor are NAD(+)-dependent DNA ligases.

L5 ANSWER 8 OF 18 MEDLINE on STN DUPLICATE 4
AN 2005446179 MEDLINE
DN PubMed ID: 15901723
TI NAD+-dependent DNA Ligase (Rv3014c) from Mycobacterium tuberculosis. Crystal structure of the adenylation domain and identification of novel inhibitors.
AU Srivastava Sandeep Kumar; Tripathi Rama Pati; Ramachandran Ravishankar
CS Division Molecular and Structural Biology, Central Drug Research Institute, Chattar Manzil, Mahatma Gandhi Marg, Lucknow-226001, India.
SO The Journal of biological chemistry, (2005 Aug 26) Vol. 280, No. 34, pp. 30273-81. Electronic Publication: 2005-05-17.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS PDB-1ZAU
EM 200510
ED Entered STN: 23 Aug 2005
Last Updated on STN: 4 Oct 2005
Entered Medline: 3 Oct 2005
AB DNA ligases utilize either ATP or NAD+ as cofactors to catalyze the formation of phosphodiester bonds in nicked DNA. Those utilizing NAD+ are
attractive drug targets because of the unique cofactor requirement for ligase activity. We report here the crystal structure of the adenylation
domain of the Mycobacterium tuberculosis NAD+-dependent ligase with bound AMP. The adenosine nucleoside moiety of AMP adopts a syn-conformation. The structure also captures a new spatial disposition between the two subdomains of the adenylation domain. Based on the crystal structure and an in-house compound library, we have identified a novel class of inhibitors for the enzyme using in silico docking calculations. The glycosyl ureide-based inhibitors were able to distinguish between NAD+- and ATP-dependent ligases as evidenced by in vitro assays using T4 ligase and human DNA ligase I. Moreover, assays involving an Escherichia coli strain harboring a temperature-sensitive ligase mutant and a ligase-deficient Salmonella typhimurium strain suggested that the bactericidal activity of the inhibitors is due to inhibition of the essential ligase enzyme. The results can be used as the basis for rational design of novel antibacterial agents.